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Studies on PHLOEM NECROSIS OF IRISH POTATO TUBERS IN WASHINGTON

AVERY E. RICH

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Studies on Phloem Necrosis of Irish Potato Tubers in Washington[1]

AVERY E. RICH[2]

INTRODUCTION

The term "phloem necrosis" as used here refers to a network of brown threadlike strands of dead phloem tissue of potato tubers originating at the stem end and extending with varying severity toward the apical end. The visibly discolored strands may be continuous or discontinuous, but they seldom extend entirely to the apical end of the tuber. They may be present in either the inner phloem, outer phloem, or both. This symptom is commonly referred to as "net necrosis" in the American literature, but this term as used by the trade and inspectors usually includes several types of discoloration. Research workers have found that this type of phloem discoloration can be produced by more than one set of factors. However, it has been shown that it frequently follows infection of the potato vines with the leaf roll virus *(Corium solani* H.).

The symptoms of chronic leaf roll (i.e. those appearing in plants grown from tubers carrying the virus from a previous season), at least as they appear in the state of Washington, are: dwarfing of the plants due to shortening of the internodes; rolling, thickening, and rigidity of the lower leaves; chlorosis of the leaves ranging from light green to yellow, sometimes accompanied by the development of a red or purple pigment; short stolons; phloem necrosis in stems and petioles; and the development of spindling sprouts, especially from those tubers showing phloem necrosis. Current-season symptoms (i.e. those developing during the same growing season in which infection occurs) include: rolling of the upper leaves, especially at the base of each leaflet, accompanied by yellowing and/or purpling; swollen nodes with zig-zag stems; and the development of phloem necrosis in stems and tubers. Phloem necrosis may develop only in the above-ground parts in those varieties showing resistance to phloem necrosis in tubers.

[1] Based on a thesis submitted to the Graduate School of The State College of Washington in partial fulfillment of the requirements for the degree of Doctor of Philosophy, May, 1950.

[2] Assistant Plant Pathologist.

Phloem necrosis of potato tubers has become a serious problem in the late crop Russet Burbank (Netted Gem) variety in Washington during the past few years. This disorder was largely responsible for the decline in production of the high quality Russet Burbank variety and an increase in the planting of White Rose, a variety somewhat inferior in quality to Russet Burbank but in which phloem necrosis is not a problem. At about the same time, a similar shift in varieties took place in Maine where growers changed from the high quality Green Mountain variety to the Katahdin, a variety with slightly lower cooking quality but rarely affected with phloem necrosis. Because the market for Washington potatoes is largely dependent on their high quality, growers hesitate to produce and market a variety of lower quality. In fact, the Russet Burbank type has become an easily recognized trademark of high quality, and any defect which detracts from its appearance or eating quality its very undesirable. Phloem necrosis usually cannot be recognized without removing the stem end of the tuber. Therefore, a very rigid inspection program is necessary in order to determine which lots are affected with this disorder. Lots which are disqualified constitute a considerable loss to the producer. Consequently an effective control program is very important.

REVIEW OF LITERATURE

Early Observations

Quanjer (104)[3] originated the term "phloem necrosis" in connection with the dead or discolored phloem tissues in the stems of potato plants affected with leaf roll. Although he did not apply this term to a similar condition in the tubers, Gilbert's work (52, 53, 54, 55, 56, 57) later showed that it would be quite applicable. W. A. Orton (97) described what appears to have been the same type of discoloration, calling it "net necrosis," a term suggested by Dr. H. W. Wollenweber. Orton also included a very clear photograph of the symptoms in Plate II, Fig. 2 of his report. He differentiated between this type of discoloration and the browning of the xylem bundles caused by *Fusarium oxysporum* Schlecht., which he referred to as "stem-end browning."

C. R. Orton (94) discussed net necrosis in his bulletin on potato diseases, but his illustration of the disease resembles

[3] Numbers within parentheses refer to "Literature Cited," p. 42.

internal rust spot[4] more closely than it does phloem necrosis. He associated the disease with certain soil conditions such as a lack of potash and dry weather.

McAlpine (80), in discussing the cause of missing hills in Australia, mentioned "thread-eye" and "spindle disease." He suggested that growers could recognize thread-eye by the occurrence of slender shoots and by a network of dark streaks in the flesh of the tuber. It is highly probable that this network of dark streaks was phloem necrosis, and that it was associated with the development of spindling sprouts.

Artschwager (4, 5, 6) made a careful histological study of potato leaf roll and phloem necrosis in American and European varieties. His description of the external symptoms of European leaf roll corresponds very closely to the disease as it occurs in this country. A microscopic study demonstrated that plants typically affected with leaf roll always showed pathological changes in the vascular tissue. The changes were more pronounced in plants which showed early and intense external symptoms. He found that primary phloem was most commonly affected; but the pericycle, fibers, and cortex often were diseased also. The phloem was rarely shrunken and completely destroyed. In the American varieties which he examined, the external symptoms tended to be less severe; and there was less correlation between external and internal symptoms than in European varieties.

Association of Tuber Phloem Necrosis With Leaf Roll

Schultz and Folsom (125) were among the first to associate tuber phloem necrosis with leaf roll. They stated that it seemed to be a symptom associated with the leaf roll disease. Experiments showed that apparently it was not associated with fungi or bacteria, but was transmissible through the tubers as are leaf roll and mosaic. In their studies, phloem necrosis was never present in lots free from leaf roll, was never more abundant than leaf roll in lots containing both diseases, and was restricted to the tubers which transmitted leaf roll. The facts that leaf roll tended to produce small tubers while phloem necrosis was frequently found in the larger ones, and that phloem necrosis often was not transmitted in the stock while the leaf roll was, might seem to discredit the relationship. These facts, however, were explained on the basis that phloem necrosis may be a temporary effect follow-

[4] See description of internal rust spot on p. 15.

ing immediately after infection of tubers by the leaf roll virus. Phloem necrosis seemed to be related to spindling-sprout also. Necrotic tubers usually tended to produce spindling-sprout, and they in turn tended to produce very weak, leaf roll plants. They found that some of the factors which tended to influence the amount of necrosis were variety, size of tubers, opportunity for leaf roll infection, and abundance of aphids.

Folsom (41) described both leaf roll and phloem necrosis as they occurred in Maine, and stated that there was much evidence to show that this type of necrosis was a part of the leaf roll syndrome. In addition to the evidence above, he noted that they both involved the same kind of internal injury—a death of the food-transporting channels.

Kasai (73) described the "shashaki" disease on potatoes in Japan, stating that it was probably identical with the leaf roll of Europe and North America. Among other symptoms of this disease he included phloem necrosis.

Gilbert (53, 54) found that net necrosis of the phloem necrosis type was a consistent current-season symptom of leaf roll, but the necrotic symptoms were not persistent in the progeny of the net-necrosis tubers. He (52) made an attempt to eliminate the confusion which existed at that time between phloem necrosis associated with leaf roll and other types of tuber necrosis such as brown fleck, internal brown spot, sprain, and streak. Some of these types of discoloration could be differentiated from phloem necrosis in one or more of the following ways: (a) they affected various internal areas of the tuber but not specifically the phloem; (b) the necrotic conditions were not progressive during storage as in the case of phloem necrosis; and (c) the causes of these necroses were not transmitted by the tubers in such a way that there were any characteristic, observable effects upon the plants grown from affected tubers. Net-necrosis tubers almost invariably produced leaf roll plants. On the other hand, tubers from leaf roll plants very rarely showed phloem necrosis. These facts indicated that phloem necrosis was a current-season or "primary" symptom of leaf roll.

Gilbert (55, 56) produced phloem necrosis under controlled conditions, thereby proving its relationship to leaf roll. He grew healthy and leaf roll plants under cages to exclude undesirable insects. Aphids of the species *Myzus persicae* (Sulz.) were colonized on leaf roll plants under cages and transferred at intervals to the foliage of healthy plants also under cages. Phloem necrosis was found in abundance in the tubers from

all the inoculated plants, while the tubers from the check cages showed no necrosis. Recently Davidson (28) reported similar results. (Schultz and Folsom [125] had suggested the above relationship from work done earlier in Maine, but had transferred their aphids from leaf roll plants grown from phloem necrosis tubers.)

Elze and Quanjer (38, 109) demonstrated that the virus of phloem necrosis of European potato varieties is identical with the American leaf roll virus, although the European varieties do not exhibit tuber phloem necrosis.

Folsom (42) described phloem necrosis and stem-end browning and made a practical distinction between the two. Ninety-nine per cent of the tubers which he considered as affected with phloem necrosis produced leaf roll plants, but only 6 per cent of the tubers showing stem-end browning produced leaf roll—a percentage comparable with that in check plants from tubers which showed no stem-end discoloration. Leaf roll infection was followed by phloem necrosis only under certain conditions—in certain varieties, in tubers produced by plants growing from healthy seed and infected with the leaf roll virus during the growing season preceding the harvest of the affected tubers, and after several weeks or months in storage. The prevention of leaf roll infection would prevent development of phloem necrosis, but the discarding of tubers showing phloem necrosis would not eliminate all leaf roll.

Effect of Environmental Factors on Development of Leaf Roll Phloem Necrosis

Environmental factors also play an important role in the development of phloem necrosis. It usually does not show up in tubers at harvest time but develops after potatoes have been stored for several weeks or months (Folsom [42], Folsom, *et al.* [49], Folsom and Rich [50]). Folsom (45) found that phloem necrosis developed most rapidly when stored at 45° to 50° F. Such development was reduced if freshly harvested potatoes were held at 70° for 60 days or at 32° to 36° for 30 to 60 days.

Ross *et al.* (122) found that there was a positive correlation between the amount of phloem necrosis and the amount of phosphorus and chloride applied in the fertilizer. Lime and organic matter increased stem-end browning but not phloem necrosis. Increasing the amount of complete fertilizer in-

creased the amount of leaf roll without a corresponding increase in the amount of phloem necrosis.

Influence of Variety on Development of Leaf Roll Phloem Necrosis

Most of the early work by Schultz, Folsom, and Gilbert was done with the Green Mountain variety. Folsom (41) noted that in Aroostook County, Maine, Irish Cobblers and Spauldings (Rose 4) showed phloem necrosis only rarely. Elze and Quanjer (38) found that the American variety, Green Mountain, developed tuber phloem necrosis while all Dutch varieties tested did not, even when they were grown side by side. According to Smith (128), Golden Wonder develops phloem necrosis in its tubers.

Maine workers (102, 103) reported that some of the newer American varieties such as Katahdin, Chippewa, Sebago, and Houma do not develop phloem necrosis in their tubers, although they are susceptible to leaf roll. Stevenson, *et al.* (132) also reported that Katahdin, Chippewa, and Sebago very rarely, if ever, show phloem necrosis in their tubers as a result of current-season infection with leaf roll virus. Stevenson (131) added Kennebec to this list, but stated that Mohawk, which was reported as resistant to phloem necrosis at first (Hardenburg and Stevenson [62]), later developed this disorder. Cassell (24) reported finding phloem necrosis in one lot each of Katahdin and Earlaine. Although he did not attempt to explain the occurrence of this type of discoloration in these varieties, it is possible that some agent other than the leaf roll virus was responsible. Sanford and Grimble (124) reported phloem necrosis in Vick's Extra Early, Carter's Early Favorite, and Russet Burbank (Netted Gem).

Ross (119) found that all Green Mountain tuber lines tested were equally susceptible to leaf roll and phloem necrosis.

Other Factors Related to Phloem Necrosis

Low temperature

Jones and his associates (71, 72) described a phloem necrosis due to frost which was not tuber transmitted. They observed that the interior vascular regions are most sensitive to this type of low temperature injury. An exposure of 2 hours at –5° C., 1 hour at –9°, or 8.5 hours at –1° produced

necrosis in some tubers. The stem end was found to be most sensitive. Either a "ring," "blotch," or "net" necrosis might be produced. Schultz and Folsom (125) also produced artificially a frost necrosis which resembled the type of phloem necrosis associated with leaf roll, by exposing tubers to low temperatures but not freezing them. Eastham (32, 33) described a vascular necrosis of potato tubers which he attributed to frost in the field. However, most of the discoloration was confined to the vascular (xylem?) ring. It is probable that this type of injury was a result of the frost killing the vines.

Folsom, *et al.* (49) stated that freezing damage sometimes resembled phloem necrosis if the freezing had not been too severe. However, it could be differentiated from the latter usually because it followed no uniform pattern, and was usually accompanied by a form of blotching.

Wright and his coworkers (136, 137, 138) studied freezing injury of potato tubers, especially as it occurred in transit. They found that the average freezing point was 29° F., but it varied with individual tubers within each variety. Symptoms vary from a ring necrosis due to mild injury, to a net-type of necrosis due to moderate injury, or a blotch type due to a more severe injury. Of course, tubers that are frozen very much break down completely.

Insect injuries

Sanford and Grimble (124) reported phloem necrosis in Netted Gems after tomato psyllids *(Paratrioza cockerelli* Sulc.) had fed on them. Seed pieces developed weak, spindly sprouts, but plants later developed normally and produced normal tubers.

Edmundson (36) attempted to produce net necrosis in potato tubers by colonizing psyllids *(Paratrioza cockerelli* Sulc.) on potato plants, but his results were negative. Snyder *et al.* (129, 130) reported a type of internal necrosis in White Rose tubers which they believed was a tuber symptom of psyllid yellows. They observed this discoloration in tubers from plants infested with large numbers of psyllids. Spindling sprout appeared to be associated with this type of injury also.

List (78), on the other hand, attributed phloem necrosis in potato tubers to feeding of the six-spotted leafhopper, *Macrosteles divisus* (Uhl.). He concluded that the discoloration was an effect of the aster yellows virus which is transmitted by the leafhopper. Manns (85) cited evidence to indicate that

leafhoppers (*Empoasca* spp.) were the cause of phloem necrosis in Delaware. He found that 50 per cent of the Green Mountain and Pontiac tubers from unsprayed plots developed this disorder. When these necrotic tubers were planted, they did not produce leaf roll plants.

Davidson (28) colonized nonviruliferous peach aphids (*Myzus persicae* Sulz.) and leaf roll-infected peach aphids on healthy potato plants. Tubers from plants on which the former had fed did not develop phloem necrosis while those from plants on which the latter had fed developed severe phloem necrosis, indicating that aphids alone were not the cause of the tuber discoloration.

Other Vascular Discolorations

McKay (83), in a study of the transmission of wilt diseases in seed potatoes in Oregon, usually found *Verticillium albo-atrum* Reinke and Berth. and *Fusarium oxysporum* Schlecht. in the stem-end vascular region of potato tubers produced by diseased plants. Their presence was usually, but not always, indicated by the characteristic discoloration of the vascular region of the stem end. *Fusarium radicicola* Wollenw. was often found in the discolored regions of the tubers, and the discoloration could not be distinguished macroscopically from that produced by the other two organisms. *F. oxysporum* produced the heaviest discoloration, while *V. albo-atrum* caused the least discoloration. *F. radicicola* was known to produce a wilt in the plants. There were several other organisms which invaded the stem end of potato tubers. Some of these were parasitic on potatoes and others were not. They were usually accompanied by a discoloration of the vascular tissues. Discoloration in the stem-end vascular region of potato tubers was not found to be a guarantee of the presence of disease-producing organisms. Forty-five per cent of the discolored tubers cultured gave organisms which cause disease in potatoes, and 55 per cent gave either no organism or miscellaneous non-pathogenic fungi.

Goss (58) likewise found that only a small percentage of the tubers showing vascular discoloration contained *Fusarium oxysporum.* A discoloration of the vascular system of both stems and tubers often developed under conditions of high temperature and low moisture, in the absence of any causal organism. Neither vascular discoloration of the tuber nor of the stem was a good index of *F. oxysporum* infection unless found in connection with a wilted plant.

Weniger (135) found that, contrary to popular belief, Fusarium wilt was found associated with less than one-half of the discolored tubers which she studied in North Dakota. In a large percentage of the tubers, blackleg bacteria were found. Both blackleg and wilt organisms were found in some, and many tubers were sterile. These discolorations were not distinguishable on the basis of color or extent.

Edson (37) also showed that vascular discoloration of the stem end of the tuber was not proof of the presence of parasitic fungi. Vascular necrosis might often arise from purely physiological causes.

Burr (20) stated that corky bacteriosis was a ring disease of the xylem vessels caused by *Bacterium suberfaciens* (Burr). It caused a browning and lignification of the vascular ring. Hopkins (66) reported the occurrence of this disease in Rhodesia also.

Folsom, *et al.* (47) reported the presence of a bacterial red-xylem disease in Maine. The surface of the stolon-scar lesion is depressed but not discolored, its interior is dark brown to black and a cavity may develop. It resembles the vascular discoloration due to herbicides, but xylem of the latter is sterile. It has been incorrectly called Fusarium wilt by inspectors, but this disease is not known to occur in Maine. The Katahdin variety is most commonly affected.

Folsom (42), in discussing stem-end browning, stated that in the broadest sense it might be considered to include all such discoloration due to fungi, bacteria, degeneration diseases, physiological and environmental causes, and unknown causes. In Maine, however, Fusarium wilt was unknown, Verticillium wilt was rare, and frost necrosis was readily recognized by the blotch-type injury or breakdown associated with it. Therefore, in that state, the term "stem-end browning" had come to mean "stem-end browning due to unknown causes."

Experiments showed that stem-end browning had no apparent effect on vine vigor or yield, and that it was not perpetuated from one generation of tubers to the next. The trouble seemed to be seasonal in any one region, regional in any one season, and not inherent in any strain or stock. Usually stem-end browning did not extend into the tubers so far, or in so many zones, as phloem necrosis. However, there may be all degrees of browning up to the stage that is severe enough to be confused with the milder stages of phloem necrosis. Since phloem necrosis in Irish Cobblers and Spauldings

(Rose 4) was rare in Aroostook County, discolorations in these varieties could be attributed generally to stem-end browning. Even in Green Mountains, stem-end browning was more common than phloem necrosis.

Folsom and Rich (50) made a study of stem-end browning as it occurs in the Green Mountain variety in Maine, and attempted to differentiate it from phloem necrosis. Neither malady was observed at harvest time, but one or both developed in certain lots in storage. Stem-end browning affected both the phloem and the xylem, but net necrosis was found only in the phloem. Stem-end browning was darker colored, occurred in fewer concentric zones, and usually did not penetrate the tuber so deeply as phloem necrosis. Phloem necrosis was correlated with leaf roll, but stem-end browning was associated with no other plant symptoms. The exact cause of stem-end browning was not known.

Rich (115) found that the addition of boron to the soil had no influence on the subsequent development of stem-end browning in the tubers. Chandler and Ross (25), using sand culture and nutrient solutions, found that a boron-deficient nutrient solution favored the development of stem-end browning.

Ross (120) found that one commercial strain of Green Mountain was more susceptible to stem-end browning than other strains, but all were equally susceptible to phloem necrosis. All Irish Cobbler lots were highly susceptible to stem-end browning. He (121) advanced the hypothesis that a virus is the cause of stem-end browning, based on transmission obtained by inarch and tuber grafts. He concluded that the Keswick strain of Green Mountain contains a virulent form of this virus, while some other strains contain either a weak strain of the virus or are free from it.

Ross *et al.* (122) found that the amount of stem-end browning was positively correlated with the amount of chloride and potassium applied in the fertilizer. Lime and organic matter increased the amount of stem-end browning. These factors were considered "contributory" rather than "causal."

C. R. Orton and Hill (95) described a new potato disease occurring in West Virginia, Maryland, and Pennsylvania. The first external symptoms of the disease were a dwarfing, paling, and upward folding of the terminal leaflets. In a few days the vines wilted and died. The vascular tissue of the entire plant, including the tubers, turned brown. One of the

characteristics of the disease was a discontinuous dendritic necrosis of the stem end of the tuber, similar to net necrosis, but less extensive in the internal phloem. These writers (95) called this disease "blue stem," and compared it with other diseases with which it might be confused. The pronounced vascular discoloration throughout the plant suggested the presence of vascular parasites such as *Bacterium solanacearum*, *Fusarium* spp. or *Verticillum*. The outstanding difference between blue stem and the above-mentioned diseases was the distribution of the necrosis in the tubers. The necrosis caused by the vascular parasites was confined almost exclusively to the vascular ring, while in the case of blue stem the cortex and pith were discolored also.

There was convincing evidence that blue stem was not a bacterial or fungus disease, including failure to isolate a pathogen after repeated attempts. Field experiments indicated that blue stem was associated with an insect vector. It first appeared in the border rows or hills of a field and spread toward the center. The disease appeared very rarely in plants protected from insects by muslin cages. Other experiments indicated that it was not transmitted through the tubers.

In recent years, the disease described in the above paragraph has been referred to as "purple-top wilt" by several workers (Brentzel [18], Decker [29], Epps [39], Younkin [139]) who have associated it with the aster yellows virus. MacLeod (79) used the term "bunch-top" for this disease or one similar to it. Menzies (86) has obtained two distinct viruses from potato plants showing purple-top symptoms. When grafted on tomato, some plants gave symptoms similar to bunch-top while others developed typical tomato big bud. Apparently, vascular discoloration of the tubers varies considerably with the variety, location, and other factors.

Folsom (44) described a disease known as "yellowtop" in Maine. In addition to the symptoms described by Orton and Hill for blue stem, he observed spindliness, stiff leaf texture, and small tubers strung along the stolons giving a "necklace" effect. While the phloem necrosis was similar to that produced by leaf roll, it developed during the growing season in the field instead of after the potatoes were placed in storage. The production of aerial tubers helped distinguish yellowtop from leaf roll. It also resembled some other virus diseases, including eastern aster yellows, purple top, witches'-broom, and apical leaf roll, as well as the current-season symptoms of leaf roll.

Sanford and Clay (123) reported a new disease of potato in Alberta which they called "purple dwarf." Symptoms include stunting, distortion, checking of apical growth, and the development of a purple hue along the margins of the leaves which are usually cupped. The phloem of the entire plant is disorganized, resulting in a brown dendritic necrosis extending the entire length of affected tubers. Perpetuation of the disease through the tubers and transmission by grafting indicate that a virus is involved. Milbrath (87) described a similar disease, occurring in Oregon, using the name "green dwarf." No mention was made of any tuber necrosis associated with it, however. Hutton and Oldaker (70) described a similar disease from Tasmania, calling it "rosette." They attributed it to a virus and noted the presence of phloem necrosis in the stems.

Milbrath and English (88) described a "late-breaking" virus disease of potatoes which was similar in many respects to purple-top wilt, yellowtop, and several other diseases. However, they found no necrosis in either the stem or the tubers.

Hilborn and Bonde (64) studied a new form of low temperature injury in potato tubers which they called "mahogany browning." Katahdins and Chippewas exposed to near freezing temperatures for an extended period of time were especially susceptible. The discoloration was not confined to the phloem. Leaf roll-infected tubers were often affected worse than healthy ones. Folsom (46) found that predisposition to this type of injury was inherited in certain varieties and seedlings.

Several workers (Hoyman [67, 68], Callbeck [23], Eastman [34], Rich [117]) have reported a discoloration of the xylem ring of potato tubers following the application of vine killers. Rich (117) reported the same type of discoloration when vines were killed by frost. It is probable that this is the same type of discoloration to which Eastman (32, 33) referred. In general, those chemicals which killed the vines most rapidly resulted in the most tuber discoloration. In nearly all cases the damage was confined to the xylem ring, and it often extended the entire length of the tuber.

Non-Vascular Tuber Discolorations

A number of different types of potato tuber discolorations have been reported which are not confined to the vascular tissues. Some of them are attributed to viruses while others

are due to soil, temperature, or unknown causes.

Barrus and Chupp (10), in their description of yellow dwarf of potatoes (a virus disease) as it occurred in New York, noted necrosis in the upper portion of the plant in the form of brown specks in the pith and cortex. They noted internal tuber necrosis as well, which appeared as rusty specks or areas surrounding the pith area, and in the cortex, being most prevalent at the middle and bud end of the tuber, and usually absent at the stem end.

Black (11), Fernow and Black (40), Muncie (92), Smith (128), Walker and Larson (134), and Holmes (65) also reported an internal tuber necrosis associated with yellow dwarf. According to Smith, and Walker and Larson, the necrotic area is not confined to the phloem but also attacks the pith and sometimes the cortex. It is easily confused with internal brown spot, but the misshapen and cracked tubers help identify it. Holmes (65) listed the virus as *Aureogenus vastans* (H.) Black. Larson (74) noted varietal differences in resistance to this disease.

Schultz and Folsom (126) described unmottled curly dwarf, another virus disease of potatoes, but apparently they did not associate internal necrosis of the tubers with it. Goss (59) noted that none of the tubers infected with the spindle tuber virus showed an internal necrosis, while frequently the unmottled curly dwarf tubers showed a light browning of the tissue either as streaks or flecks. In some tubers the discoloration resembled that associated with Fusarium wilt, while others were similar to the type of necrosis associated with leaf roll. Folsom (44) also reported observing an internal tuber necrosis associated with unmottled curly dwarf. He noted that the discoloration was in the pith, similar to that found in yellow dwarf tubers and that it was different from the "net" necrosis found in leaf roll and "yellowtop" tubers. Holmes (65) classified the unmottled curly dwarf (*Acrogenus solani* var. *severus* H.) as a strain of the spindle tuber virus (*Acrogenus solani* H.).

Atanasoff (8, 9) claimed that in Holland net necrosis is a tuber symptom of aucuba mosaic but not of leaf roll. He also stated that net necrosis has no relation to leaf roll. However, his illustrations do not show typical phloem necrosis, but a blotchy type of necrosis scattered through the tuber. It is possible that he was dealing with pseudonetnecrosis, described below. However, several workers (Clinch, *et al.* [27], Smith [128], Holmes [65]) have reported a necrosis of the pith and

cortex of tubers of certain varieties when infected with the aucuba mosaic virus *(Marmor aucuba* H.). Dykstra (31) described a new disease, Canada streak, which he considered to be a strain of aucuba mosaic (designated as virus G by Clinch, *et al.* [*27*]). This disease caused an internal necrosis of potato tubers which developed in storage. Among the varieties affected were Irish Cobbler, Sebago, President, Arran Victory, Chippewa, Green Mountain, and Bliss Triumph. This disease had not been reported in the United States and had not become serious in Canada. Aucuba mosaic produced no tuber symptoms in the American varieties with which he was working.

Oswald (98) described a disease of White Rose potatoes which produced severe leaf, stem, and tuber necrosis. Primary symptoms consisted mainly of internal necrotic browning of veins, petioles, and stems, followed by collapse of the entire plant in severe cases. Affected leaves showed either chlorotic or necrotic spots. The top leaflets became rugose and curled downward. Tuber necrosis occurred just under the skin, later becoming sunken and corky, and sometimes spreading throughout the entire tuber. Internal tuber necrosis in the second generation was less severe. He (99) later reported that the virus was a strain of the alfalfa-mosaic virus, and called it the tuber-necrosis virus.

Quanjer, Thung, and Elze (111, 112) described "pseudonetnecrosis" of the potato. In some strains of the variety Red Star, an internal parenchyma necrosis was found as numerous brown spots inside and outside the vascular ring; and it was transmitted by the seed tubers to the progeny. It spread to neighboring plants in the field, but had no foliage symptoms combined with it. It was transmitted experimentally by the aphid *Myzus persicae* from Red Star to Duke of York potato plants. Potato varieties differed in susceptibility. Paul Kruger (President) was very susceptible, showing the necrotic spots on the outside skin. The necrosis developed in storage more quickly at high than at low temperatures. On account of its late development, the transmission by seed tubers might often be overlooked.

Quanjer (108) distinguished between phloem necrosis which is restricted to the sieve tubes and companion cells, and phloem parenchyma necrosis (pseudonetnecrosis) in which the spots are in the storage parenchyma of the tubers only.

Clinch, *et al.* (*27*) suggested that pseudonetnecrosis and tuber blotch were apparently caused by the same virus (virus

F), which produced a tuber necrosis in the parenchymatous cells of both the cortex and pith. Smith (128) lists them as synonyms under *Solanum* virus 8.

Dykstra (31) also concluded that the viruses (designated as F by Clinch, *et al.* [27]) causing pseudonetnecrosis and tuber blotch were identical. A blotchy type of necrosis frequently developed in the parenchyma tissue of several varieties, including President, Bliss Triumph, Irish Cobbler, Katahdin, and Chippewa.

Clinch (26) recognized two types of tuber blotch—"true" tuber blotch, caused by virus F, and virulent tuber blotch, caused by a more virulent strain of virus F. Cross inoculation experiments proved that infection with one strain made plants immune to the other, thus establishing close relationship.

Another group of diseases has been described (1, 2, 3, 7, 19, 20, 21, 22, 60, 61, 66, 75, 76, 81, 89, 90, 93, 101, 107, 110, 133, 140) which has caused considerable confusion in a number of different countries. Among the names which have been used for one or more types of these diseases are Kringerigheid, Eisenfleckigkeit, Buntfleckigkeit, Buntwerden, maladie des taches en couronne, Pfropfenbildung, sprain, spraing, corky bacteriosis, internal rust spot, internal brown spot, internal brown fleck, and corky ring spot. Apparently at least two distinct types of symptoms are involved. In one case, rusty or dark brown spots are scattered more or less throughout the central portion of the tuber. These spots are variable in size, and most workers have found no parasitic organism associated with them. The other type is characterized by more or less concentric arcs or rings of discolored corky tissue on the surface or underneath the skin, and usually having a lenticel or skin injury for a focal point. However, the above names have been used interchangeably so much that it is impossible to assign one set of names to one type of injury and reserve the other set of names for the other disease.

Due to the confusion resulting from the loose use of the term "sprain" and other terms, McIntosh (81) offered the following differentiating descriptions of the discolorations:

> (a) *Internal Rust Spot*—The flesh of the affected tubers shows rusty, brown markings, varying in size from mere specks to large blotches 1 cm. or more in diameter and irregularly distributed. In severe cases, cavities occur in the necrotic tissue. The precise cause of the disease is not yet satisfactorily determined. The disease is common in Scotland.

(b) *Sprain*—The cut surface of affected tubers shows arcs, or curved splashes, of dead tissue, arranged more or less parallel to, or concentric with, one another. These arcs often appear to have as their focal point either a lenticel or a slight abrasion of the skin of the tuber. Here again there is no satisfactory explanation of the cause of the disease, which does not appear to be very common in Scotland.

There were several other diseases which could not be described so readily. Some of them were caused by viruses, some were caused by mineral deficiencies, and the cause of others was not known.

Appel (3) described and illustrated the two types of disease defined above by McIntosh. He stated that the rust-spot type ("Eisen" or "Bluntfleckigkeit") is nonparasitic, and is associated with light, sandy soils or loam soils with a gravelly subsoil. The concentric-ring type (called "Pfropfenbildung" in Germany and "Kringerigheid" in Holland) appears to be soil-transmitted and is considered a virus disease by some investigators. It may be a virus disease that is expressed only under certain soil conditions.

It seems probable that the first type is synonymous with internal brown spot (12, 114, 127) in America, while the latter type corresponds to the American disease, corky ring-spot (35, 113).

History of Phloem Necrosis in the Pacific Northwest

Heald (63), in describing "net necrosis" in Washington, wrote:

> Little is known in regard to the cause of this disease but the few observations point to unfavorable soil conditions as one of the inciting factors . . . tests indicate that the trouble is not transmitted by the use of affected seed . . . net necrosis is closely related to internal brown spot . . . The same corrective measures are recommended.

He stated that the leaf roll was tuber-transmitted, but there was no visible change in the tuber. Spindling sprout was not associated with leaf roll or net necrosis but was thought to be due to storage or growing conditions.

McKay (82) described "net necrosis" as:

> characterized by the production of an extensive network of small brown strands of discolored tissue extending

through the interior of the potato tuber tissue though occurring more abundantly in the tissues near the surface of the tubers. In some cases this condition is evidently brought about by chilling or frosting, but it is possible that other conditions may produce it also. The disease is not inherited . . . Affected tubers should be avoided as seed, due to the chance of confusing them with potatoes affected with wilt.

Link (77) noted that in the Northwest, leaf roll was most common in northeastern Washington, northern Idaho, and western Montana, where it affected primarily the Russet Burbank. He reported that "It has increased decidedly during the period 1919-1922 and is spreading." Aphids were first found in potato fields in 1922 after 3 years' search. Virus symptoms were often masked by the high temperatures, low humidities, and intense sunlight of this region. No mention was made of net necrosis in connection with leaf roll.

Zundel (141) commented on "net necrosis" as follows:

> This is a condition of the tuber characterized by a network of long brown strands of dead tissue extending throughout the tuber but more abundant near the outer edge . . . It is perhaps not caused by any one factor . . . Recent work in the east has associated this as one symptom of leaf roll, while work in Wisconsin has associated it with one type of frost necrosis. A third cause may be soil and moisture conditions similar to those that cause internal brown spot, namely: an insufficient supply of water and lack of potash and perhaps phosphorus.

McKay and Dykstra (84) gave a description of "net necrosis" which is very similar to that given earlier by McKay (82). They stated that experimental work in the East shows that it is closely associated with leaf roll and is apparently a symptom of it. They also recorded the fact that this type of symptom has been found in tubers affected with witches'-broom, and that similar symptoms might be brought about by chilling or frosting the tubers.

Huber (69) reported that phloem necrosis in Snohomish and Skagit counties (in Washington) ranged from a trace to 30 per cent in the 1938 crop and from a trace to 42 per cent in the 1939 crop. Eighty-four out of 201 hills showed one or more necrotic tubers. Affected tubers usually produced spindling sprouts. The necrotic tubers produced plants with symptoms similar to leaf roll, while the non-necrotic ones

from the same hills produced apparently healthy vines. The first symptoms of current-season infection resembled Rhizoctonia, with the exception of aerial tuber formation. Only those vines which showed symptoms produced necrotic tubers.

Morris and Afanasiev (91) stated that spindling sprout is not a separate disease but is a symptom of witches'-broom and leaf roll. According to these workers, "net necrosis" may be a symptom of frost injury, Fusarium wilt, leaf roll, or unfavorable growing conditions. They suggested that excessive heat in storage could also cause discoloration in the tubers.

Boyle made several surveys of potato diseases affecting the 1943 and 1944 crops in Washington and Oregon (13, 14, 15, 16). He (16) made the following comment on the 1943 crop in Oregon:

> Although by chance no severe cases of leaf roll were noted in the limited observation of fields, several cases were encountered during bin inspection later. The entire crop from certain fields was not marketable because of inability to separate necrotic tubers from others.

In another report (13) he wrote:

> Net necrosis (virus) caused complete loss of certain lots of potatoes. The tubers appeared sound but there was no possible way to grade these and obtain the tolerance allowed for any market grade. These lots were of the Netted Gem variety. According to the observation of certain growers the prevalence of this disease was correlated with seed source. Whether the current infection was from unobserved infected seed pieces or from unsuspected weed hosts is a question for further observation and study.

In discussing potato storage diseases in western Washington, Boyle (14) stated that phloem necrosis was reported to have caused heavy losses and that certain producers claimed that this defect results in more loss than any other.

Boyle and Blodgett (17) reported that phloem necrosis (virus) was common in the Yakima Valley and Kittitas County in the 1944 crop, but to a much smaller degree than in the same area the previous year. The same disorder was also reported at Moses Lake.

According to Dodge and Anderson (30), leaf roll was unimportant in Washington until 1938 when it became destructive in the Ellensburg area. It seemed to spread rapidly, and by 1943 it was a serious problem in the production of Netted Gems in all of the irrigated areas. Current-season infection

was common. In summarizing the effects of leaf roll, they stated that tubers from leaf roll plants frequently showed a phloem necrosis, but this symptom was not dependable; they may develop necrosis in storage even though none was evident at harvest.

Foster (51) attributed phloem necrosis to various causes. According to him it is a form of injury similar to frost necrosis, but occurs in tubers which have not been exposed to frost. In some cases it seems to be due to causes similar to those producing internal brown spot; in other cases it seems to be associated with leaf roll.

Oswald and Kendrick (100) reported the occurrence of phloem necrosis and stem-end browning in Netted Gem potatoes grown in California. Phloem necrosis was associated with leaf roll, but stem-end browning was not.

Rich (115) reported that infestation with viruliferous aphids early in the season produced a higher percentage of necrotic tubers than when infestation occurred late in the season. The plants which were infested early also showed a greater reduction in yield.

Rich and Locke (118) examined twenty different varieties and two unnamed seedlings grown at three different locations for the development of internal discoloration in the tubers. Varieties which showed no phloem necrosis include White Rose, Katahdin, Calrose, Essex, Placid, Glenmeer, and seedling X1276-185. Varieties which showed more than a trace of phloem necrosis include Russet Burbank, Warba, Red Warba, Ontario, Cayuga, Chenango, Snowdrift, and Ashworth. The last variety exhibited by far the most discoloration, with 45.7 per cent of the tubers affected.

MATERIALS AND METHODS

The Russet Burbank (Netted Gem) variety of potato was used in all experimental work except where otherwise indicated. All greenhouse work was conducted in the Plant Pathology greenhouse at The State College of Washington, Pullman. Field experiments were conducted at the Plant Pathology experimental plots at Pullman, at the Irrigation Experiment Station at Prosser, at the Northwestern Washington Experiment Station at Mt. Vernon, and on private farms at various locations in the state. The work falls naturally into four phases, which will be treated separately as follows: (a) severity of phloem necrosis in Washington, (b) primary cause of phloem necrosis in Washington, (c) varietal and

clonal reaction to phloem necrosis, and (d) testing of possible control measures.

SEVERITY OF PHLOEM NECROSIS IN WASHINGTON

Surveys

In the fall of 1947, the writer observed that phloem necrosis was present in some lots of potatoes at harvest time. This is not in agreement with the literature or with conditions as observed in Maine. Consequently, a survey was initiated in order to determine how widespread and how serious this type of necrosis was at that time of year. Table 1 shows that

Table 1. Farm Survey Showing Percentage of Phloem Necrosis Occurring in Russet Burbank Potato Tubers at Harvest Time, 1947.

Field Number	Location	Percentage Phloem Necrosis	Field Number	Location	Percentage Phloem Necrosis
1	Moses Lake	3	16	Ellensburg	1
2	Moses Lake	5	17	Ellensburg	0
3	Moses Lake	3	18	Ellensburg	2
4	Moses Lake	7	19	Ellensburg	3
5	Moses Lake	7	20	Ellensburg	3
6	Moses Lake	5	21	Ellensburg	5
7	Moses Lake	10	22	Wapato	2
8	Moses Lake	5	23	Wapato	4
9	Moses Lake	2	24	Wapato	0
10	Moses Lake	8	25	Wapato	1
11	Prosser	3	26	Mt. Vernon	0
12	Prosser	2	27	Lynden	0
13	Prosser	3	28	Lynden	0
14	Prosser	1	29	Pullman	6
15	Prosser	0	30	Pullman	2

this disorder was most prevalent at Moses Lake, where it occurred in all lots examined. The portion of tubers discolored ranged from 2 to 10 per cent at this location. Phloem necrosis occurring at harvest time varied from 0 to 5 per cent in the Ellensburg area, from 0 to 4 per cent in the Wapato area, and from 1 to 4 per cent in the Prosser area. Two lots at Pullman showed 2 and 6 per cent, respectively. No discoloration was observed in samples from Mt. Vernon and Lynden in western Washington.

In order to further determine the severity of phloem necrosis in Washington, another survey covering the 1947, 1948,

Questionnaire on Net Necrosis

1. Was net necrosis a problem in the 194__ crop of Netted Gem potatoes?_____ (No, slight, or severe)
2. Was it a problem in early potatoes?_____
3. Was it a problem in late potatoes?_____
4. Was it a problem at harvest time?_____
5. Was it a problem in stored potatoes?_____
6. Net necrosis was a grade-limiting factor in approximately what percentage of the Netted Gems in your area?____%

Name_____
(Horticultural Inspector)

Fig. 1. Sample questionnaire used to obtain information on extent of phloem necrosis in potatoes in Washington during 1947, 1948, and 1949.

and 1949 crops was made. Questionnaires (Fig. 1) were sent to the horticultural inspectors in the state, who inspected all commercial potatoes at the time they were graded and shipped. This survey showed that necrosis was often a grade-limiting factor in the late-crop Russet Burbanks. It varied considerably from one locality to another in any given year and from one year to another in the same locality (Fig. 2). Potatoes in the Moses Lake area were most seriously affected in 1947, phloem necrosis being serious enough to reduce the grade in from 10 to 15 per cent of the late crop potatoes. In 1949, the Ellensburg area was most severely affected. Phloem necrosis was a grade-limiting factor in about 40 per cent of the crop in the latter area.

Development in Storage

In order to ascertain the rate of development of phloem necrosis in storage, potato tubers from lots which showed a certain amount of discoloration at harvest time were divided into samples of 100 tubers each, as nearly uniform as possible in size, maturity, opportunity for insect infestation, and leaf roll infection. Four samples of 100 tubers each were examined for the presence of phloem necrosis. Each tuber was weighed and its length measured, after which a thin slice was removed from the stem end. If phloem necrosis was visible at the cut surface, thin slices were removed successively until the dis-

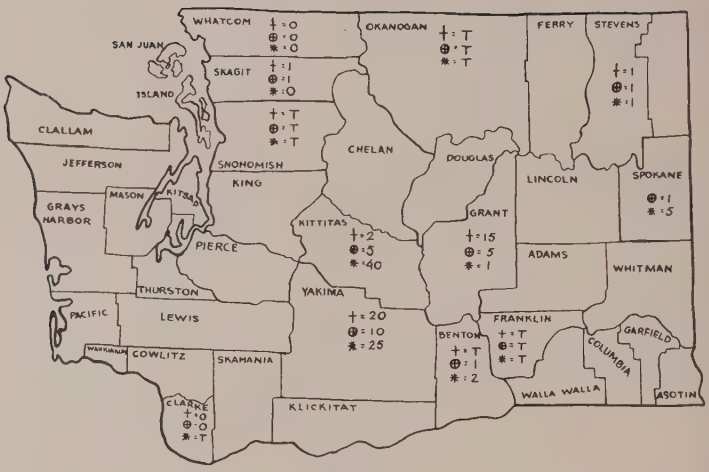

Fig. 2. Percentage of potato tubers (by counties) in which phloem necrosis was a grade-limiting factor, 1947-1949.

coloration was no longer evident in the remaining portion of the tuber. Then the clear-fleshed portion of the tuber was reweighed and remeasured. From these data it was possible to calculate the percentage of the tubers discolored and the depth of penetration of the necrotic tissue into each tuber.[5] The remaining samples were placed in a controlled temperature room at 42° F. At monthly intervals, four 100-tuber samples were removed from storage and examined according to the method just described. Table 2 gives the percentage of necrotic tubers found each month and the average depth of penetration of the discoloration into the tubers. It will be noted that the percentage of discolored tubers increased during the first 2 or 3 months in storage, or until January. The depth of penetration, on the other hand, remained relatively constant during this period but increased during the latter part of the storage period (January to March). A possible ex-

[5] The weight data are presented and discussed elsewhere. See p. 26.

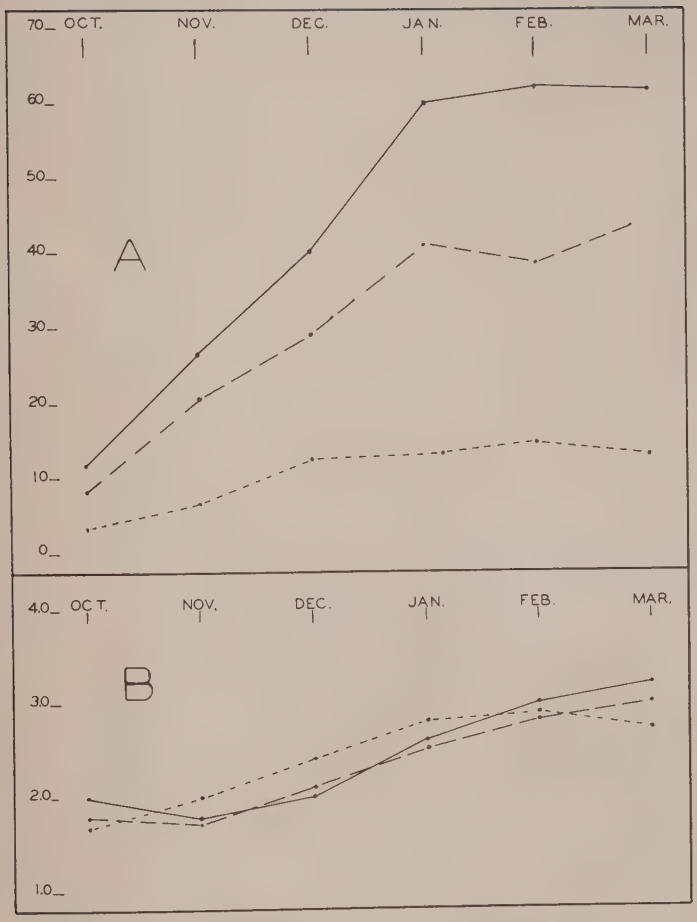

Fig. 3. Effect of length of storage on development of phloem necrosis in Russet Burbank potato tubers. "A" shows percentage of tubers affected when samples were removed at monthly intervals and examined. "B" shows the depth of penetration expressed in centimeters for those potato tubers.

Table 2. Effect of Length of Storage on Development of Phloem Necrosis in Russet Burbank Potato Tubers—1948 Crop.

Source	Month of Examination											
	October		November		December		January		February		March	
	Percentage of Tubers Affected	Depth (cm.) Penetrated	Percentage of Tubers Affected	Depth (cm.) Penetrated	Percentage of Tubers Affected	Depth (cm.) Penetrated	Percentage of Tubers Affected	Depth (cm.) Penetrated	Percentage of Tubers Affected	Depth (cm.) Penetrated	Percentage of Tubers Affected	Depth (cm.) Penetrated
Prosser	12.0	2.0	26.5	1.8	40.2	2.0	60.0	2.6	62.7	3.0	62.0	3.2
Pullman	8.5	1.8	20.7	1.7	28.5	2.1	41.2	2.5	38.7	2.8	44.0	3.0
Mt. Vernon	3.2	1.7	6.5	2.0	12.2	2.4	12.7	2.8	14.0	2.9	12.7	2.7
Mean	7.9	1.8	17.9	1.8	27.0	2.2	38.0	2.6	38.5	2.9	39.6	3.0

Standard error of mean for percentage of tubers affected = 5.06.
Standard error of mean for depth of penetration = 0.103 cm.

planation for the fact that the average depth of penetration did not increase during the early part of the storage period might be that discoloration was just beginning to develop in some of the tubers and had not penetrated very deeply, thus reducing the average depth of penetration. However, by January most of the tubers which would ever show necrosis had developed at least a limited amount of discoloration which progressed in storage during the next few months.

Another experiment was set up to determine the effect of storage temperature on the development of phloem necrosis in the tubers. Samples were prepared as in the preceding experiment and stored at six different temperatures ranging from 32° F. to 80° F. At the end of 2 months, the potatoes were removed from storage and examined for phloem necrosis according to the method just described. The results are reported in Table 3. The amount of necrosis found in

Table 3. Percentage of Russet Burbank Potato Tubers Showing Phloem Necrosis at Harvest and After Storage for Two Months at Various Temperatures.

Source	Storage Temperature						
	At Harvest	32° F.	42° F.	50° F.	60° F.	70° F.	80° F.
Prosser	23	25	44	45	49	47	32
Pullman	15	21	52	48	51	49	28
Mt. Vernon	12	16	39	42	45	44	21
Mean*	17	22	45	45	48	47	27

* Standard error of mean = 1.71.

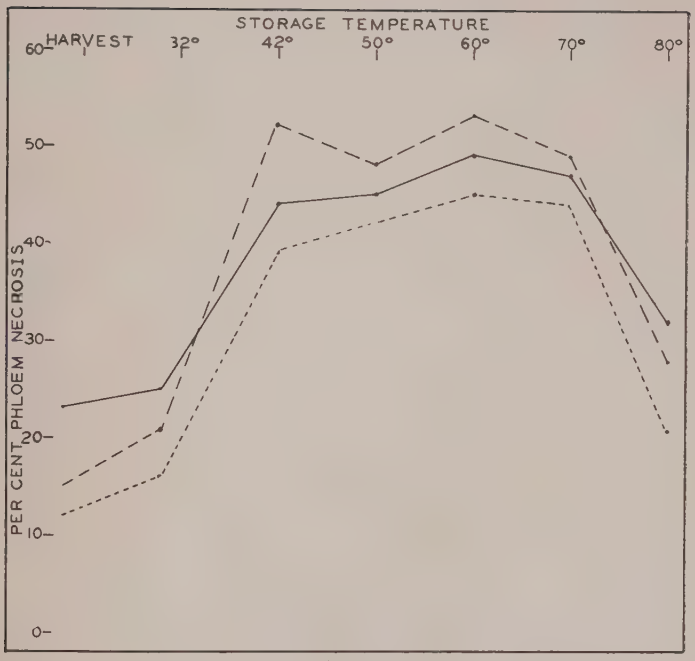

FIG. 4

LEGEND:

PROSSER
PULLMAN
MT. VERNON

Fig. 4. Percentage of Russet Burbank potato tubers showing phloem necrosis at harvest time and after storage for 2 months at various temperatures.

potato tubers stored at 32° was only slightly more than was present in comparable samples at harvest time. Tubers stored at approximately 42°, 50°, 60°, and 70° developed an appreciably greater amount of discoloration than developed at 32°, but there was very little difference in the amount developing at these four temperatures. However, samples stored around 80° developed more necrosis than at 32° but less than at the four intermediate temperatures. These results do not agree

exactly with those obtained with the Green Mountain variety in Maine. (See section on review of literature, p. 5.

By dividing the tubers from the above samples into weight classes and calculating the percentage of necrotic tubers in each class, it was possible to show an association between the amount of necrosis with tuber size, as shown in Table 4.

Table 4. Percentage of Russet Burbank Potato Tubers in Various Size Classes Developing Phloem Necrosis.

Source	Tuber Weight Classes				
	2-4 oz.	4-6 oz.	6-8 oz.	8-10 oz.	Over 10 oz.
Pullman	25.5	40.7	55.2	50.0	52.9
Prosser	15.9	21.1	29.9	36.7	47.5
Moses Lake	22.0	33.2	38.2	37.5	48.9

These results show that the smallest tubers had the lowest incidence of disease and that it became progressively worse as the size of tubers increased. These results agree with those reported for Green Mountain in Maine.

Effect on Yield

An experiment was conducted during the growing season of 1949 to determine the effect of phloem necrosis on yield. The stem ends and bud ends of necrotic tubers grown in 1948 were planted in randomized blocks, along with stem ends and bud ends of non-necrotic leaf roll tubers from the same lot. Stem-end sets from leaf roll-free tubers, and from tubers grown from stock which contracted leaf roll in 1946 and 1947 were also included. The average stand and yields are reported in Table 5. A very marked and highly significant reduction in

Table 5. Effect of Necrotic and Non-necrotic Russet Burbank Leaf Roll Seed Tubers on Stand and Yield.

Leaf roll Content	Year of Infection	Necrotic Tubers	Source of Set	Percentage Stand	Yield in Tons/Acre
—	—	—	Stem end	94	10.15
+	1948	+	Stem end	42	1.33
+	1948	+	Bud end	46	1.54
+	1948	—	Stem end	46	1.64
+	1948	—	Bud end	44	1.61
+	1947	—	Stem end	52	2.10
+	1946	—	Stem end	56	2.27

Standard error of a mean difference, Healthy vs. Diseased = 0.77.
Standard error of a mean difference, between Diseased = 0.20.

stand resulted in all cases where leaf roll-infected sets were used regardless of whether or not they showed phloem necrosis. The sets from the stem end of necrotic tubers produced the poorest stand and lowest yield of any lot. However, the differences were not statistically significant at the 19:1 probability level in most cases.

All yields were extremely low, with the exception of the plots planted with healthy sets. The growing season was unusually dry, and many of the cut sets remained dormant in the soil until the fall rains came. Then some of them commenced to sprout. However, since these young plants were very small at harvest time (many of them having failed to even emerge from the soil and none of them having set tubers), they were not counted when stand counts were made. These results would indicate that phloem necrosis as well as probably some invisible injury to phloem in leaf roll tubers may be partially to blame for poor stands in potato fields when leaf roll-free seed is not used.

Time of Inoculation

The effect of time of inoculation with the leaf roll virus on subsequent development of phloem necrosis in the tubers was studied by means of a cage experiment. Cloth cages (2′x2′x2′) were constructed, and four plants were grown under each cage from leaf roll-free seed potatoes. Viruliferous aphids reared on leaf roll potato plants were introduced into four of the cages July 4th, about 1 month after planting. The plants in four more cages were infested with similar viruliferous aphids 2 weeks later. This procedure was continued throughout the growing season. In the fall, each hill of potatoes was harvested separately and placed in storage for 2 months at a temperature favorable for development of phloem necrosis. Then each tuber was examined for the presence of this type of discoloration. The results are summarized in Table 6. The percentage of necrotic tubers ranged from 79 for the tubers produced from those plants which were infested first, to 49 for the tubers produced by those plants infested near the end of the growing season. The non-infested plants produced non-necrotic tubers. The lowest yield was produced by those plants infested earliest in the season. The yields gradually increased as the date of infestation was delayed, and the plants infested last produced almost as much as the non-infested plants. The distance which the necrosis penetrated into the tubers varied very little, regardless of date of

Table. 6. Effect of Time of Leaf Roll Infection on Amount and Severity of Phloem Necrosis and on Yield of Russet Burbank Potatoes.

Date of Infestation	Percentage Necrotic Tubers[a]	Depth (cm.) Discolored	Yield in Tons/Acre[b]
July 4	79	1.7	7.83
July 18	60	2.0	8.41
Aug. 1	69	2.8	8.99
Aug. 14	67	2.3	8.99
Aug. 28	49	2.3	10.44
Check	0	0	10.73

[a] Least difference required for significance (19:1 probability level) = 8.4 per cent.

[b] Least difference required for significance (19:1 probability level) = 0.48 tons per acre.

infestation. The fact that the depth of discoloration was shallowest in the tubers produced by the plants which were infected first might possibly be accounted for by the smaller size of the tubers.

Research workers and growers have sometimes expressed the opinion that necrosis probably resulted from leaf roll infection occurring late in the season. However, these results indicate that early infection is even more serious than late infection from the standpoint of both tuber necrosis and reduction in yield.

PRIMARY CAUSE OF PHLOEM NECROSIS IN WASHINGTON

It has already been pointed out that a number of different factors have been claimed to cause phloem necrosis. Therefore, it seemed desirable to determine the primary cause or causes of the disorder in this state.

In 1947, necrotic potato tubers were collected from as many different sources as were then available, and they were indexed for virus content in the greenhouse. All tubers diagnosed as being affected with phloem necrosis produced either leaf roll plants or no plants at all. Some of the non-necrotic tubers from the same lots produced normal plants while others produced leaf roll plants, the proportion depending on the amount of leaf roll in the stock. Several split hills were found where some tubers were infected while other tubers from the same hill were healthy. In fact, a very few split tubers were found where certain eyes of a tuber produced leaf roll plants while others from the same tuber produced plants which ap-

peared normal. In order to obtain further proof as to the cause of phloem necrosis, potato plants were grown under cloth cages as described under the previous heading. Green peach aphids *(Myzus persicae)* were reared on leaf roll plants in the greenhouse and were introduced into certain of the cages at intervals during the summer. Some of the tubers produced by the plants under each cage infested in this manner showed phloem necrosis. On the other hand, none of the tubers produced by the non-infested plants showed this type of internal browning. (See Table 6.)

A similar method was used to produce phloem necrosis in the greenhouse. By placing viruliferous aphids on healthy, caged plants, growing the plants to maturity in pots, and holding the tubers thus produced for several weeks in storage, it was possible to produce phloem necrosis in some of the tubers. In order to determine whether or not aphids affect the development of phloem necrosis other than as vectors of the leaf roll virus, healthy plants were grafted with leaf roll scions and caged to exclude insects. The tubers produced by these plants were examined for necrosis after they were stored for several weeks, and they were found to contain phloem necrosis which was comparable in amount and severity to that produced by aphid inoculation (Table 7). Therefore, it seems evident that the only role played by the insect is solely that of a virus vector.

Table 7. Comparison Between Numbers of Russet Burbank Potato Tubers Developing Phloem Necrosis Following Aphid Inoculation and Graft Inoculation with the Leaf Roll Virus.

Type of Inoculation					
Aphid			Graft		
Plant Number	Necrotic Tubers	Non-necrotic Tubers	Plant Number	Necrotic Tubers	Non-necrotic Tubers
1	2	3	1	2	3
2	1	3	2	1	2
3	2	3	3	2	2
4	1	2	4	0	5
5	0	6	5	1	1
6	2	4	6	1	2
7	1	3	7	1	2
8	1	2	8	1	3
9	1	1	9	1	3
10	2	2	10	1	3
11	1	2			
12	0	6			

VARIETAL AND CLONAL REACTION TO PHLOEM NECROSIS

Considerable work has been done in Washington and other states in connection with varietal resistance of potatoes to leaf roll. Observations have also been made in regard to phloem necrosis development in some of the varieties. However, very few careful studies have been made of the varietal differences in potatoes with respect to the development of phloem necrosis in the tubers. Therefore, a study was made of this phase of the problem in conjunction with the variety testing program[6] already under way in the state. Twenty named varieties and two unnamed, pedigreed seedlings were grown in randomized blocks at three locations in 1948. Rows of Russet Burbank infected with leaf roll were interspersed among the varieties to serve as a source of inoculum. Approximately 30 medium-sized tubers of each variety were selected at random from each block at harvest time and were placed in storage for several months.

In the spring, the amount of phloem necrosis in each lot was determined by removing the stem end of each tuber and counting the number of affected tubers. The percentage of affected tubers found at each location is shown in Table 8. Tubers from the Prosser plots showed the least phloem damage, and Pullman lots usually showed the most. The Ashworth variety showed by far the most discoloration at all three locations. Other varieties which showed an appreciable amount of injury at one or more locations include Russet Burbank, Warba, Red Warba, Ontario, Cayuga, and Chenango. White Rose, Katahdin, Calrose, Essex, Placid, Glenmeer, and seedling X1276-185 were entirely free from this defect.

In 1949, sixteen varieties and one seedling selection were included in a similar test. At harvest time, 50 medium-sized tubers were chosen at random from each plot and placed in storage for 2 months or more before cutting. The results are summarized in Table 9. As in the 1948 crop, the Ashworth variety was most seriously affected. Madison, which was not included in the 1948 test, was affected almost as seriously as Ashworth, however. Russet Burbank was damaged considerably more than any other variety in the test, with the exception of these two. Other varieties which showed 4 per cent or more necrosis at one or more locations are Russet, Sebago, Warba, LaSoda, and Chenango. Varieties which remained free

[6] Under the direction of Dr. S. B. Locke and Dr. C. L. Vincent.

Table 8. Incidence of Phloem Necrosis in Tubers of Twenty-two Potato Varieties Grown at Three Locations in 1948.

Variety	Location						Total Three Locations	
	Pullman		Prosser		Mt. Vernon			
	Total Tubers	Percentage Phloem Necrosis	Total Tubers	Percentage Phloem Necrosis	Total Tubers	Percentage Phloem Necrosis	Total Tubers	Percentage Phloem Necrosis
Russet Burbank	120	0.8	117	2.6	127	9.4	364	4.4
White Rose	120	0	119	0	119	0	358	0
Katahdin	119	0	121	0	127	0	367	0
Warba	113	10.6	113	0	125	7.2	351	6.0
Red Warba	120	8.3	119	0	123	0	362	2.8
Ontario	120	0	120	0	129	9.3	369	3.3
Mesaba	120	0	116	0.9	126	0	362	0.3
Norkota	120	2.5	121	0.8	123	0	364	1.1
Cayuga	115	10.4	121	0.8	127	5.5	363	5.5
Menominee	121	4.1	122	0	125	0	368	1.4
Calrose	119	0	114	0	125	0	358	0
Kennebec	66	1.5	117	0	91	0	274	0.4
Essex	121	0	120	0	126	0	367	0
Chenango	120	1.7	123	0	123	6.5	366	2.7
Placid	120	0	124	0	124	0	368	0
Snowdrift	121	1.7	113	0	126	4.8	360	2.2
Cortland	120	1.7	119	0	124	0	363	0.6
Harford	120	0.8	115	0	126	0	361	0.3
Glenmeer	.....	.....	113	0	126	0	239	0
Ashworth	120	70.8	116	19.8	121	45.5	357	45.7
X1276-185	98	0	122	0	125	0	345	0
C.S.6316	97	0	121	0.8	123	0	341	0.3

from this disorder are White Rose, Glenmeer, Calrose, and X1276-185.

There is some evidence that the large amount of phloem necrosis found in the Ashworth and Madison varieties is not associated with current-season leaf roll infection but may be related to some other factor or factors. When cuttings from affected tubers were planted in the greenhouse, very poor stands frequently resulted, especially in the case of Madison. In many instances, sets from tubers showing phloem necrosis produced apparently healthy plants also.

In another test, Green Mountain was compared with Russet Burbank grown at Prosser. The Green Mountain variety appeared to be affected considerably more than Russet Burbank, both as to number of tubers showing phloem necrosis and the severity of damage to individual tubers, as shown in Table 10. It is possible that the greater depth of penetration in the case of the Green Mountain is associated with the depression at the point of stem-end attachment which is typical of this variety.

Table 9. Frequency of Phloem Necrosis in Tubers of Sixteen Varieties and One Unnamed Seedling Grown at Three Locations in 1949.

	Percentage of Necrotic Tubers*			
	Location			Average 3 Locations
	Prosser	Mt. Vernon	Pullman	
Russet Burbank	17.0	23.0	9.0	16.3
White Rose	0	0	0	0
Sebago	1.5	3.0	0.5	1.7
Russet Sebago	1.5	5.5	0	2.3
Ashworth	49.5	56.5	28.5	44.8
Warba	0	5.0	1.0	2.0
Red Warba	0.5	0.5	0	0.3
Kennebec	0.5	1.0	1.5	1.0
Madison	45.0	46.0	34.0	41.7
Ontario	2.5	2.0	1.0	1.8
Essex	0	0.5	2.5	1.0
Glenmeer	0	0	0	0
LaSoda	3.0	4.5	2.0	3.2
Calrose	0	0	0	0
Chenango	1.5	4.0	2.0	2.5
Snowdrift	0	3.0	2.5	1.8
X1276-185	0	0	0	0
L.S.D. (5%)	2.4	1.6	2.0	

* Based on examination of 200 tubers from each location, 50 chosen at random from each of 4 replicates, except for Kennebec at Prosser and Mt. Vernon where fewer than 200 were available.

Table 10. Comparison Between Phloem Necrosis Injury to Green Mountain and Russet Burbank Tubers Grown at Prosser, 1949.

Variety	Percentage Phloem Necrosis	Average Depth of Penetration	Typical Color
Green Mountain	20.2	2.8 cm.	Dark Brown
Russet Burbank	8.8	2.2 cm.	Light Brown

In another experiment, a search was made for a strain or clonal line of Russet Burbank that would be less susceptible to phloem necrosis of the tubers. Six different lots were obtained from Canadian and Idaho growers who had maintained their own seed stocks for at least 10 years. These were planted in randomized blocks at Prosser and Pullman. The percentage of necrotic tubers which developed in each lot at each location is shown in Table 11. Although some differences were

Table 11. Phloem Necrosis Development in Different Clonal Lines of Russet Burbank Potatoes at Two Locations.

Clonal Line	Percentage Phloem Necrosis	
	Prosser	Pullman
1	9.6	5.0
2	6.2	2.3
3	11.2	1.4
4	10.0	8.2
5	5.8	6.0
6	9.8	2.2

found at each location, they were not consistent. For example, lot 4, which showed the most phloem necrosis at Prosser, showed the least at Pullman. These results indicate that none of the lots tested was superior to the others from the standpoint of development of phloem necrosis.

TESTING OF POSSIBLE CONTROL MEASURES

Prolonging Dormant Period

All varieties included in the 1948 variety trials (Table 8) were treated with methyl ester of naphthaleneacetic acid (Barsprout) shortly after harvest to determine varietal differences in effectiveness of this material to inhibit sprout development.[7] In the spring, after completion of data on sprout development, the potatoes were cut and data on phloem necrosis were obtained. These results are recorded in Table 12. Apparently, this treatment had no influence on the development of phloem necrosis.

In another test, 3 lots of Russet Burbank were treated with the same sprout-inhibiting compound, and three comparable

[7] This phase of the experiment was conducted by Dr. C. L. Vincent who kindly permitted Dr. S. B. Locke and the writer to examine the tubers in the spring for development of phloem necrosis.

Table 12. Effect of a Sprout Inhibitor on Development of Phloem Necrosis in the Tubers of Twenty Potato Varieties and Two Seedlings.

Variety	Percentage Phloem Necrosis	
	Barsprout*	No Treatment
Calrose	0	0
Cayuga	2.5	7.1
Norkota	3.4	4.8
Mesaba	0	0
Harford	0	0
Netted Gem	3.9	2.9
Essex	0	0
Katahdin	1.2	0
X1276-185	0	0
Ashworth	22.2	17.2
CS6316	0	0
Snowdrift	0	0
White Rose	0	0
Menominee	0	0
Warba	0	1.5
Glenmeer	0	0.6
Kennebec	0	0
Chenango	0	0
Cortland	0	0
Red Warba	0	1.7
Placid	0	0
Ontario	1.6	0

* Methyl ester of naphthaleneacetic acid.

lots were left untreated. Then a treated and an untreated lot were stored at 42°, 60°, and 80° F., respectively. When the potatoes were cut, there was no appreciable difference between the treated and the comparable untreated check at any of the temperatures.

Isolated Seed Plots

In the spring of 1948, foundation seed potatoes were planted in isolated plots at a number of different locations in the state, as shown in Table 13. All plots except those at Prosser and Pullman were isolated from potatoes carrying the leaf roll virus. However, the Mt. Vernon and Colville plots were not so well isolated as the remaining plots because there were other potatoes about 1,000 feet from them. Inspection of all plots during the summer indicated that the seed was free from the leaf roll virus. Samples were harvested at intervals during the late summer and fall in order to determine when and how much disease spread took place. During the winter the samples were examined for phloem necrosis and were in-

Table 13. Location of Isolated Seed Plots, Spread of Leaf Roll, and Development of Tuber Phloem Necrosis in 1948.

Location	Date Planted	Date of Harvest	Percentage Leaf roll	Percentage Necrosis
Coupeville	Apr. 17	July 14	0	0
Coupeville	Apr. 17	Aug. 8	0	0
Coupeville	Apr. 17	Sept. 1	0	0
Colville	Apr. 26	July 28	0	0
Colville	Apr. 26	Aug. 18	0	0
Colville	Apr. 26	Sept. 13	0	0
Colville	June 11	Aug. 18	0	0
Colville	June 11	Sept. 13	0	0
Colville	June 11	Oct. 2	2.4	0
Mt. Vernon	May 9	Aug. 7	0	0
Mt. Vernon	May 9	Sept. 2	2.1	0
Spokane	May 12	July 29	0	0
Spokane	May 12	Aug. 18	0	0
Spokane	June 10	Aug. 18	0	0
Spokane	June 10	Sept. 14	0	0
Spokane	June 10	Oct. 3	0	0
Port Angeles	May 19	Aug. 8	0	0
Port Angeles	May 19	Sept. 1	0	0
Lynden	June 7	Aug. 7	0	0
Lynden	June 7	Sept. 2	0	0
Pullman*	June 9	Sept. 5	28.3	15
Pullman*	June 9	Sept. 24	41.2	28
Prosser*	June 18	Sept. 10	28.7	11
Prosser*	June 18	Oct. 17	49.6	36

* Pullman and Prosser plots were not isolated from other potatoes.

dexed in the greenhouse for leaf roll. The results are recorded in Table 13. A small amount of leaf roll was found in the plants produced by the late-harvested samples from Mt. Vernon and Colville, but no necrosis was observed in the tubers. Considerable phloem necrosis was found in the samples from both Pullman and Prosser, and the tubers produced a high percentage of leaf roll plants. At both locations the samples harvested late in the season showed the most disease spread. All samples from the other locations were free from phloem necrosis and leaf roll.

The above results indicated that leaf roll and phloem necrosis of potato tubers could be controlled satisfactorily in many parts of Washington by planting leaf roll-free seed potatoes in fields which are well isolated from potatoes carrying this disease. Supporting evidence was obtained in 1947 with the White Rose variety. Isolated plots were located at Prosser, Vancouver, Long Beach, Forks, Joyce, and Whidbey Island. According to the results obtained from indexing samples from the various plots, no leaf roll infection took place

at any of the locations, with the exception of Prosser. This isolated plot contracted 3 per cent leaf roll late in the season. As this variety does not develop phloem necrosis in its tubers, Russet Burbanks were used in subsequent tests.

In 1949, it was decided to carry out a more severe test of various localities to determine which ones were most favorable and which were least favorable for leaf roll spread and possible development of tuber necrosis. Therefore, potato plots were planted with leaf roll-free seed at the locations shown in Table 14. However, at each location one row was

Table 14. Location of Test Plots, Spread of Leaf Roll, and Development of Tuber Phloem Necrosis in 1949.

Location	Date Planted	Date of Harvest	Percentage Leaf-roll	Percentage Necrosis
Long Beach 1	Apr. 19	July 19	0	0
Long Beach 1	Apr. 19	Sept. 15	0	0
Long Beach 2	Apr. 19	July 19	0	0
Long Beach 2	Apr. 19	Sept. 15	0	0
Vancouver	Apr. 20	July 19	0	0
Vancouver	Apr. 20	Sept. 14	3.3	2.0
Spokane	Apr. 25	July 26	0	0
Spokane	Apr. 25	Aug. 24	0	0
Spokane	Apr. 25	Sept. 23	2.2	1.0
Spokane	June 24	Aug. 24	0	0
Spokane	June 24	Sept. 23	2.5	1.0
Colville	Apr. 26	July 26	0	0
Colville	Apr. 26	Aug. 23	0	0
Colville	Apr. 26	Sept. 22	4.8	2.0
Colville	June 24	Aug. 23	1.5	0
Colville	June 24	Sept. 22	5.0	2.0
Mt. Vernon	May 6	Aug. 4	0	0
Mt. Vernon	May 6	Sept. 16	20.0	11.0
Lynden	May 8	Aug. 4	0	0
Lynden	May 8	Sept. 16	0	0
Port Angeles	May 20	Aug. 3	0	0
Port Angeles	May 20	Sept. 15	0	0
Pullman	May 27	Aug. 17	1.0	0
Prosser	June 2	Aug. 18	77.8	6.0
Prosser	June 2	Sept. 13	95.6	10.0
Prosser	June 2	Oct. 1	96.3	17.0

planted with potatoes known to be infected with leaf roll so that the plants produced by them could serve as a source of inoculum for the others. Tuber samples were harvested at intervals during the late summer and fall. During the winter, the tubers were examined for phloem necrosis and were indexed to determine the amount of leaf roll pick-up. Again, under the 1949 growing conditions, very little leaf roll spread took place at any of the locations tested except Prosser, even

when a row of leaf roll plants existed as a source of inoculum. From the experience of seed potato growers in the state in years past, this lack of leaf roll spread is not typical. However, it appears reasonable to assume that if leaf roll-free seed is planted in isolated plots, leaf roll spread and subsequent development of phloem necrosis in the tubers would be held to a minimum.

Early vs. Late Planting

General observations indicated that phloem necrosis was more severe in late potatoes than in potatoes grown for the early market. The questionnaire survey (Fig. 1) supported this observation. None of the inspectors reported that necrosis was a problem in the early crop, but most of them who are located in the commercial potato producing areas indicate that it was at least a minor problem in the late crop. There are several factors which might account for this difference in severity of phloem necrosis. The variety grown, stage of maturity at harvest time, soil and air temperature at harvest time, and whether or not potatoes are placed in storage are some of the factors that may influence the development of this type of tuber injury. Other important factors may be the population and movement of aphids which carry the leaf roll virus and the part which the early crop may play as a source of inoculum for the late crop. When the early crop matures or is harvested green, the aphids move to the late crop and carry with them any virus which they have picked up.

An attempt was made in the Spokane and Colville areas to measure the effect of planting date on phloem necrosis development. Potatoes were planted as early in the spring as weather permitted and again in June, the usual planting date for these areas (Tables 13 and 14). However, the results show very little leaf roll spread and practically no phloem necrosis in the tubers, regardless of planting date.

Early Harvesting vs. Harvesting at Maturity

As already indicated and shown in Tables 13 and 14, samples were harvested on two or more different dates usually about 3 or 4 weeks apart, toward the end of the growing season. At all locations where leaf roll and phloem necrosis were found, these disorders grew progressively more severe in the samples harvested toward the end of the growing season.

Effect of Various Fertilizers on Development of Phloem Necrosis

A fertilizer experiment was conducted[8] at Kittitas in 1949. Four levels of nitrogen, three levels of phosphorus, two levels of potash, and two forms of sulfur were applied in fourteen various combinations. At harvest time, the potatoes from each plot were dug and weighed separately, after which they were placed in storage. During the latter part of December, the tubers were cut and information was obtained on the prevalence of phloem necrosis in each lot, as reported in Table 15.

Table 15. Effect of Various Fertilizer Treatments on Subsequent Development of Phloem Necrosis in the Potato Tubers.

Treatment (Pounds Per Acre)				Percentage Phloem Necrosis[d]
N	P_2O_5	K_2O	S	
0	0	0	0	5.2
80	0	0	0	1.1
40	40	0	0	1.3
0	40	60	0	3.1
40	80	0	0	2.6
40	40	60	0	1.8
40[a]	40[a]	60[a]	0	4.3
80	40	60	0	2.0
120	40	60	0	6.2
40	80	60	0	4.0
80	80	60	0	2.0
120	80	60	0	2.2
80	40	60	400[b]	0.8
80	40	60	400[c]	3.2

[a] Broadcast before planting.
[b] As elemental sulfur.
[c] As gypsum.
[d] Least difference required for significance (19:1 probability level) = 3.9 per cent.

However, results were quite inconsistent, and there was very little, if any, correlation between fertilizer treatments and the subsequent development of necrosis in the tubers produced.

Use of Aphicides

Attempts to control leaf roll by the suppression of aphids with insecticides have been made by numerous investigators in years past. However, the reduction of aphid populations to a very low level was not always followed by a correspondingly low incidence of leaf roll and phloem necrosis in the

[8] The fertilizer experiment was conducted by T. L. Jackson of the Agronomy Department.

crop. During the past few years, DDT and other new insecticides have been used experimentally and commercially for the control of aphids. However, reports on their effectiveness in controlling virus diseases have been variable. In 1949, an insect control experiment was conducted in central Washington. Four applications of ten different insecticides were applied with a ground duster.[9] Aphid counts were made at six different dates during the growing season. Table 16 shows the

Table 16. Effect of Various Aphicides Applied to Russet Burbank Potato Plants on Subsequent Development of Phloem Necrosis in Their Tubers.

Treatment	Aphids*		Percentage Phloem Necrosis	
	Winged	Wingless	Fall	Spring**
1. 5% DDT	1257	12,590	16.7	10.2
2. 5% DDT + 2% oil	1275	15,696	13.7	8.6
3. 3% DDT + 2% oil	1211	13,467	14.5	8.4
4. 4% DDT + 80% sulfur	796	8,179	9.0	5.4
5. 33.3% sodium fluoaluminate	1306	14,585	18.0	13.0
6. 33.3% sodium fluoaluminate + 0.5% parathion	1311	10,037	11.2	10.4
7. 5% DDT + 75% sulfur	630	5,957	7.5	8.7
8. 0.5% parathion	966	8,730	8.7	11.8
9. 0.5% parathion + 5% DDT	986	7,756	5.7	7.6
10. 1.0% parathion	1358	10,610	10.0	9.1

* Represents total aphids counted on 2,400 compound leaves per treatment. (Counts of 400 leaves per treatment were made on 6 different dates.)

** The potatoes examined in the spring were size B tubers. This may account for the smaller percentage of necrotic tubers then than was found in the fall.

list of insecticides tried, a summary of aphid counts following their use, and percentage of tubers with phloem necrosis in samples harvested from the various plots. These results indicate that DDT plus sulfur (treatments 4 and 7) and a combination of DDT and parathion (treatment 9) gave sufficient control of aphids to reduce the amount of phloem necrosis which later developed in the tubers. A study of the aphid counts for each date suggests that sulfur acted as a repellent during period of migration and that parathion was effective against colonization and build-up of large populations on the plants. This would suggest the possibility that in the not-too-distant future a new insecticide or a new formulation of some of the present insecticides may be developed which will give sufficient control of aphids to reduce the leaf roll spread and

[9] This experiment was conducted by B. J. Landis and coworkers on the Schmoe Farm near Harrah. These data are used with his permission.

consequent phloem necrosis of the tubers to a minimum. However, as aphids move about rather freely, such an insecticide program would probably need to be adopted on a community-wide basis.

SUMMARY

A fairly complete review of literature on phloem necrosis and related problems is presented. An attempt has been made to clarify some of the confusion which exists in the literature. Surveys showed that phloem necrosis of potato tubers occurs rather frequently at harvest time in Washington, and the damage becomes progressively more severe in storage.

Storage experiments indicated that phloem necrosis developed more rapidly at storage temperatures between 42° and 70° F. than at 32° or 80° F. The total number of affected tubers appears to increase during the first 2 or 3 months in storage. After that time, the total number of affected tubers does not increase, but the extent of damage within the affected tubers continues to increase.

When necrotic tubers are used for seed, a marked reduction in stand and yield results as compared with healthy seed. The stand and yield were also slightly poorer than those produced by leaf roll seed tubers which did not show visible phloem necrosis.

The time of infestation with viruliferous aphids appeared to have some effect on yield and development of phloem necrosis. Plants infested early in the season produced the lowest yields and greatest amount of phloem necrosis, while plants inoculated late in the growing season produced the highest yields and the lowest percentage of tubers exhibiting phloem necrosis.

Phloem necrosis occurring in Russet Burbank tubers in Washington during the period of this study was associated closely with infection with the leaf roll virus. This conclusion is based on the following evidence: (a) phloem necrosis tubers from various locations in the state all produced leaf roll plants when planted, while those which did not show phloem necrosis gave rise to both leaf roll and healthy plants; (b) plants artificially infested with leaf roll-viruliferous aphids produced tubers 49 to 79 per cent of which showed phloem necrosis; (c) plants grafted with leaf roll scions produced tubers which developed phloem necrosis in comparable amounts to those produced by plants infested with viruliferous aphids.

A study of all the varieties included in the 1948 and 1949 variety trials indicated some marked differences in tendency to develop phloem necrosis when exposed to possible leaf roll infection at three widely separated areas in the state. Ashworth was most severely affected both years. Madison was included in the 1949 variety trials and was almost as severely affected as Ashworth. However, there is some evidence that the necrosis exhibited by these two varieties is not due entirely to leaf roll. Other varieties which showed an appreciable amount of phloem injury one year or both include Green Mountain, Russet Burbank, Warba, Red Warba, Ontario, Cayuga, Chenango, Russet Sebago, and LaSoda. Varieties which remained free from this defect were White Rose, Katahdin, Calrose, Glenmeer, Placid, and seedling X1276-185. Some of the last named remained relatively free from leaf roll also. Six different clonal lines of Russet Burbank were tested for possible differences in resistance to phloem necrosis, but no clear-cut, consistent differences could be distinguished.

An attempt was made to find some practical control measure for phloem necrosis besides the use of resistant varieties. Potatoes were examined which had been treated with methyl ester of naphthaleneacetic acid to prevent breaking dormancy, but the treatment had no apparent influence on the development of this disorder. Planting leaf roll-free seed in isolated plots was very effective in controlling leaf roll and phloem necrosis in nearly every instance by reducing spread of the leaf roll virus. Phloem necrosis was less of a problem in early planted potatoes than in those which were planted late. Early harvesting, before the potatoes were mature, also appeared to reduce the amount of discoloration in some instances. The various fertilizer treatments which were tried had very little, if any, influence on the severity of phloem necrosis. The use of a combination DDT-sulfur dust or combined DDT-parathion dust appeared to give a certain amount of control of phloem necrosis in the crop produced by the dusted plants. The reduction in tuber necrosis was apparently associated with aphid control on the plants.

In conclusion, then, phloem necrosis in Washington is closely correlated with current-season spread of leaf roll. It is a serious problem in the Russet Burbank variety, but is no problem in White Rose and several other newer and less popular varieties. Control measures include planting leaf roll-free seed in isolated plots, the adoption of an adequate aphid control program, or the use of resistant varieties.

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